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NIGERIA.

ANNUAL REPORT

ON THE

MEDICAL RESEARCH INSTITUTE

FOR THE YEAR

1925.

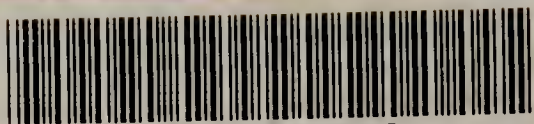


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APPENDIX A.

ANNUAL REPORT OF THE MEDICAL RESEARCH
INSTITUTE, 1925,

BY

ANDREW CONNAL, M.D., D.P.H., D.T.M., AND H.

INTRODUCTORY.

The Director has been on duty throughout the year as has been also the honorary Entomologist.

Dr. H. Morrison who was acting as Assistant Bacteriologist proceeded on leave on 27th March.

Mr. E. F. Hines, Laboratory Assistant, went on leave on 23rd May and returned to duty on 22nd October.

Dr. E. C. Smith took up the appointment of Assistant Bacteriologist on 23rd July and immediately commenced an investigation of the skin conditions prevalent in Lagos.

Mr. Hobson, Government Analyst, returned from leave on 5th February.

It is pleasant to record that the assumption of duty by Dr. G. G. Butler in October, as Pathologist at Lagos Hospital, freed the Research Institute of practically all routine work, and considerable progress has already been made in research.

In June the West African Yellow Fever Commission, of the International Health Board, commenced its labours. Relations with these workers have been pleasant and mutually helpful.

PLAGUE.

Plague in its several forms has not disappeared from Lagos although the number of its victims is considerably lessened. Small extensions of the disease have taken place during the year but not farther than Abeokuta by rail, or than the neighbourhood of Ijebu-Ode by lagoon.

Such outbreaks as did occur beyond Lagos, were restricted in area, involved few cases and were promptly and efficiently dealt with. The clinical material sent to the Institute is treated shortly under the headings of "smears," "histological" and "rodents." There was one instructive series of cases, however, which illustrates the difficulty of diagnosis. A patient was admitted to the African Hospital in July, suffering from pneumonia. The sputum was examined but plague bacilli were not found. The patient died and a native nurse who had been in attendance on him contracted pneumonia very shortly thereafter. Again in this case *B. pestis* could not be distinguished in the sputum. This nurse died and a second native nurse who had looked after his confrere contracted pneumonia also. The sputum of this third case was seen by several observers who were all agreed that *B. pestis* was not distinguishable in the stained smear. At the post-mortem examination of the third case, two guinea-pigs were inoculated on a scarified area of the abdomen with material from the lung. Stained smears from these organs showed a large number of various bacteria without any typical bi-polar staining bacilli. However, by the second day both pigs had developed buboes and both were dead on the fifth day with an acute plague infection, the only organisms present in the lesions being typical *B. pestis*. It is clear, therefore that a form of pneumonic plague occurs, in which the sputum is the typical rusty viscid one of ordinary pneumococcal pleuro-pneumonia, and in which the plague bacilli are so scanty or so altered in appearance and so mixed with other organisms that the only reliable means of diagnosis is by inoculation of a susceptible laboratory animal.

The main object of this section is to continue the recording of the results of direct inoculation of plague from guinea-pig to guinea-pig without the intermediary of the flea or of artificial culture media.

The Report of 1924 ended with Guinea-pigs A.A., Y. and Z.

Guinea-pig A.A. was the fifth animal in a direct infection from a human bubo, the bacilli in which were mostly coccoid. The transmission in each instance was made from material taken from a bubo at an interval of from one to twelve hours after death.

In order to save repetition the results are tabulated.

Table (1) shows the results from Guinea-pig A.

It will be seen that the infection died out in the thirteenth pig of the series, during a period of eighty-four days. The first animal, guinea-pig A. died in five days. The succeeding three survived seven to nine days, the fifth and sixth died in four days, the seventh in five days, and then as the table shows there are the suggestive periods of 8, 7, 4, 3, 4, 7 and 9 days.

Table (3) deals with the findings in the series beginning again from Guinea-pig A. but transmitted from that animal to Monkey C. Guinea-pig D, therefore, which heads the list was inoculated from the mixed juices of spleen, lungs, and bronchial glands of Monkey C. The sequence is a long one, comprising forty-four guinea-pigs and it continued from 30th November, 1924 until 11th July, 1925, a period of two hundred and twenty-three days. The longest survival apart from the last two guinea-pigs was nine days, and the shortest was two days. Three to seven days, however, were the most common duration.

There were two side-issues from this series. The first was from Guinea-pig P.A. in which there was a purulent condition of the bronchial glands. Guinea-pig T.A. was inoculated therefrom. It died in three days. Two small inflamed glands at the site of inoculation were the only macroscopic signs post-mortem. *B. pestis* was numerous in smears from the glands, but was not seen in the smears of the organs. Guinea-pig V.A. was inoculated from Guinea-pig T.A. but on its death five days later no signs of plague were found. The other side-issue was from Guinea-pig Y.C. This guinea-pig developed a bubo rapidly but showed no other signs of illness. After nineteen days the bubo was aspirated and the pus rubbed on a scarified area on the abdomen of Guinea-pig Z.C. From this another series was continued the details of which are to be seen in Table (2).

It has to be noted that instead of using the bubo as the source of the infective material, Guinea-pig G.D. was inoculated from the spleen of Guinea-pig F.D. In this series it will be observed that in a big proportion the lungs were affected.

These three tables show the results of plague infection in guinea-pigs in a direct line from 22nd November until 18th September, a period of two hundred and ninety-eight days.

There remains one series, as set out in Table (4).

It begins with Guinea-pig G. which was inoculated on 30th November from the contents of a human bubo, and it covers a period of one hundred and fifty-four days.

Several general conclusions can be arrived at, after reviewing these data. Rarely did the infection terminate fatally in less than three days, at the stage of an acutely inflamed bubo, and when it did happen the toxæmia must have been profound. Most of the deaths took place in from four to six days when the bubo had become hæmorrhagic and friable, with small beads of pus. In the earlier stages of this condition of the bubo, the spleen is usually finely speckled, *i.e.* with minute abscesses. In the later stages the spleen practically always shows speckling but of a coarser nature, *i.e.* larger abscesses, and in some cases the liver shows the same appearance. When the bubo had become definitely broken down and purulent it was the rule to find abscesses in both the spleen and the liver. Still later when the contents of the bubo have become thickened and caseous, the lungs may also show abscess formation.

TABLE (1).—INOCULATIONS FROM GUINEA-PIG A. TO GUINEA-PIG DB.

Guinea-Pig.	Inoculated.	Died.	Bubo.	Spleen.	Liver.	Lungs.	Heart.	Period lived.
A.	22-11-24	27-11-24	Hæmorrhagic ++ +	Speckled + + +	Speckled + + +	Congested, Pleuritis +	Petechiae +	5 days
C.	27-11-24	6-12-24	Caseous + + +	do. + + +	do. +	Abscess rt. lung + +	Pericarditis +	9 "
K.	6-12-24	14-12-24	do. +	do. + + +	Friable +	Congested +	... +	8 "
T.E.	14-12-24	21-12-24	do. + +	do. + + +	Speckled + + +	Speckled + + +	... +	7 "
X.	21-12-24	25-12-24	Hæmorrhagic +	do. + + +	Congested -	Congested -	... -	4 "
AA.	25-12-24	29-12-24	Purulent + +	do. + + +	Speckled + + +	do. + +	... +	4 "
D.A.	29-12-24	3-1-25	Hæmorrhagic + +	do. +	Friable -	do. -	... -	5 "
F.A.	3-1-25	11-1-25	Caseous +	do. +	Congested -	Exudate, rt. pleura -	... -	8 "
JA.	11-1-25	18-1-25	Hæmorrhagic + + +	do. + + +	Speckled + + +	Speckled + + +	Pericarditis -	7 "
OA.	18-1-25	22-1-25	do. + + +	do. + + +	Friable +	Congested -	... -	4 "
RA.	22-1-25	25-1-25	do. + + +	Congested -	do. -	do. -	... -	3 "
UA.	25-1-25	29-1-25	do. + + +	do. -	do. -	do. -	... -	4 "
AB.	29-1-25	5-2-25	do. +	do. -	do. -	do. -	... -	7 "
DB.	5-2-25	14-2-25	nil.	nil.	nil.	nil.	... -	9 "

TABLE (2).—INOCULATIONS FROM GUINEA-PIG ZC. TO GUINEA-PIG LD.

ZC.	8-7-25	16-7-25	Hæmorrhagic + +	Speckled +	Speckled +	Speckled + + +	...	8 days
BD.	16-7-25	23-7-25	do. + +	do. + +	do. +	Congested -	...	7 "
CD.	23-7-25	28-7-25	do. +	do. + +	Friable + +	do. + +	...	5 "
DD.	28-7-25	1-8-25	do. +	do. + +	Congested + + +	Broncho-pneumonia + + +	...	4 "
ED.	1-8-25	7-8-25	Caseous + +	do. + + +	Speckled + + +	Congested + + +	...	6 "
FD.	7-8-25	12-8-25	do. + + +	do. + + +	do. + + +	Intense Congestion + + +	...	5 "
GD.	12-8-25	18-8-25	Hæmorrhagic + +	do. +	Congested +	Congested +	...	6 "
HD.	18-8-25	24-8-25	Caseous + +	do. + +	Speckled + + +	Speckled + + +	...	6 "
ID.	24-8-25	28-8-25	Hæmorrhagic +	do. + + +	Congested + + +	Slight Congestion + + +	...	4 "
JD.	28-8-25	1-9-25	do. +	Congested + + +	do. + + +	Broncho-pneumonia + + +	...	4 "
KD.	1-9-25	18-9-25	Caseous +	Speckled. +	Speckled +	Speckled +	...	17 "
LD.	18-9-25	...	nil.	nil.	nil.	nil.

The signs + and - denote the finding or not finding of B. pestis in smears.

TABLE (3).—INOCULATIONS FROM GUINEA-PIG D. TO AD.

Guinea-Pig.	Inoculated.	Died.	Bubo.	Spleen.	Liver.	Lungs.	Heart.	Period lived.
D	30-11-24	6-12-24	Purulent ++	Speckled +++	Speckled +++	Congested +	Pericarditis	6 days.
L	6-12-24	15-12-24	" +	" +++	Friable ++	" +	"	9 "
S	15-12-24	22-12-24	Caseous +++	" +++	Speckled +++	Speckled +++	"	7 "
Y	22-12-24	26-12-24	Hæmorrhagic +	" +	Congested +	Pneumonia Rt. +	"	4 "
BA	26-12-24	2-1-25	Caseous +++	" +	"	Congested +	"	7 "
EA	2-1-25	6-1-25	Purulent +	Congested -	" -	Slightly Congested	"	(killed).
HA	6-1-25	12-1-25	Hæmorrhagic +	Speckled -	" -	"	"	4 "
KA	12-1-25	16-1-25	" ++	Congested +	" +	"	"	4 "
MA	16-1-25	19-1-25	" ++	" -	"	"	"	3 "
PA	19-1-25	22-1-25	" ++	Speckled +++	Friable -	Congested + Bronchial glands enlarged	"	
SA	22-1-25	25-1-25	Hæmorrhagic ++	Congested ++	Friable ++	Congested ++	"	3 "
YA	25-1-25	28-1-25	" ++	" ++	" +	" slightly	"	3 "
XA	28-1-25	2-2-25	Purulent ++	Speckled +	" -	"	"	3 "
BB	2-2-25	9-2-25	Hæmorrhagic ++	" ++	" +	"	"	5 "
EB	9-2-25	15-2-25	" +	" ++	Congested ++	Speckled ++	"	7 "
HB	15-2-25	18-2-25	" ++	Congested +	" +	"	"	6 "
JB	18-2-25	20-2-25	" ++	" +	" -	"	"	3 "
KB	20-2-25	25-2-25	" ++	Speckled +	" -	" slightly	Pericarditis	2 "
NB	25-2-25	1-3-25	" ++	Enlarged ++	" -	"	"	5 "
PB	25-2-25	4-3-25	" ++	Congested ++	" +	"	"	4 "
RB	1-3-25	4-3-25	" ++	" +	Pale +	"	"	3 "
RB	4-3-25	6-3-25	" ++	"	" -	"	"	2 "
TB	6-3-25	9-3-25	Congested ++	" slightly -	Dark red -	Pale -	"	2 "
VB	9-3-25	13-3-25	Hæmorrhagic ++	Speckled -	Congested -	Congested -	Pericarditis	2 "
XB	13-3-25	15-3-25	Congested +	Congested -	"	" slightly	"	4 "
ZB	15-3-25	19-3-25	Hæmorrhagic +	" ++	" +	"	"	4 "
BC	19-3-25	25-3-25	Caseous +	Speckled +	Friable +	Speckled ++	"	6 "
DC	25-3-25	30-3-25	" ++	Enlarged ++	Congested ++	Congested +	"	5 "
FC	30-3-25	3-4-25	Hæmorrhagic	Congested ++	"	" slightly	"	4 "
HC	3-4-25	8-4-25	" ++	Speckled ++	"	"	"	5 "
JC	8-4-25	12-4-25	" ++	Finely speckled ++	Deeply congested ++	"	"	4 "
KC	12-4-25	17-4-25	" ++	Speckled ++	Congested +	" slightly	"	5 "
MC	17-4-25	22-4-25	" ++	" ++	Friable +	"	"	5 "
NC	22-4-25	28-1-25	" +	" ++	Congested ++	"	"	6 "
OC	28-4-25	4-5-25	Caseous ++	" +	"	" slightly	"	6 "
QC	4-5-25	11-5-25	" +	" -	Speckled -	Speckled -	"	7 "
RC	11-5-25	17-5-25	Friable +	" ++	Congested +	Congested slightly	"	6 "
SC	17-5-25	23-5-25	Caseous +	" ++	"	"	"	6 "
TC	23-5-25	27-5-25	Purulent +	" +	"	"	"	4 "
UC	27-5-25	3-6-25	Caseous ++	" +	"	"	"	7 "
VC	3-6-25	10-6-25	" ++	" +	"	"	"	7 "
WC	10-6-25	14-6-25	Purulent +	" +	"	"	"	4 "
XC	14-6-25	19-6-25	Caseous +	" ++	"	"	"	5 "
YC	19-6-25	11-7-25	" -	" Nodules -	Swollen pale +	Hepatisation ++	"	22 "
AD	11-7-25	3-8-25	Nil -	Nil -	Congested Nil	Pale -	Nil	23 "

TABLE (4).—INOCULATIONS FROM GUINEA-PIG G. TO PC.

Guinea-Pig.	Inoculated.	Died.	Bubo.	Spleen.	Liver.	Lungs.	Heart.	Period lived.
G.	30-11-24	9-12-24	Caseous ++ +	Speckled ++ +	Speckled +	Intensely congested ++ +	—	9 days (killed).
O.	9-12-24	16-12-24	do. +	do. +	do. +	Congested +	...	7 do.
V.	16-12-24	24-12-24	do. +	Congested +	Congested +	do. +	...	8 do.
Z.	24-12-24	28-12-24	Purulent ++ +	Speckled +	Friable —	do. —	...	4 do.
OA.	28-12-24	4-1-25	Caseous ++ +	do. +	do. +	do. +	...	7 do.
GA.	4-1-25	10-1-25	Hæmorrhagic +	do. +	Congested —	do. —	...	6 do.
IA.	10-1-25	14-1-25	do. ++ +	Congested —	do. —	do. —	...	4 do.
LA.	14-1-25	17-1-25	do. +	Speckled +	do. —	do. —	...	3 do.
NA.	17-1-25	21-1-25	do. +	Congested —	do. —	do. —	...	4 do.
QA.	21-1-25	25-1-25	do. ++ +	Speckled +	Friable —	do. —	...	4 do.
WA.	25-1-25	29-1-25	do. ++ +	do. ++ +	do. ++ +	do. +	...	4 do.
ZA.	29-1-25	3-2-25	do. +	do. ++ +	Enlarged +	do. +, Bronchial glands inflamed +	...	5 do.
OB.	3-2-25	9-2-25	do. +	do. ++ +	Congested +	Congested +	...	6 do.
FB.	9-2-25	14-2-25	do. ++ +	Congested ++ +	do. +	do. +	...	5 do.
GB.	14-2-25	17-2-25	do. ++ +	Speckled +	do. —	do. +	...	3 do.
IB.	17-2-25	21-2-25	do. +	do. ++ +	do. +	Slightly congested —	...	4 do.
LB.	21-2-25	24-2-25	do. ++ +	Congested —	do. —	Congested —	...	3 do.
MB.	24-2-25	26-2-25	Acutely inflamed ++ +	do. —	do. —	do. —	...	2 do.
OB.	26-2-25	1-3-25	Hæmorrhagic ++ +	Speckled +	do. —	do. —	...	3 do.
QB.	1-3-25	4-3-25	do. ++ +	Congested —	do. —	do. —	...	3 do.
SB.	4-3-25	8-3-25	do. +	do. ++ +	do. +	do. +	...	4 do.
UB.	8-3-25	11-3-25	Acutely inflamed ++ +	do. +	do. +	do. +	...	3 do.
WB.	11-3-25	14-3-25	Hæmorrhagic ++ +	do. —	do. —	do. —	...	3 do.
YB.	14-3-25	16-3-25	Acutely inflamed ++ +	do. —	do. —	do. —	...	2 do.
AC.	16-3-25	20-3-26	Hæmorrhagic ++ +	do. ++ +	do. +	do. +	...	4 do.
CC.	20-3-25	28-3-25	Caseous ++ +	Speckled +	Speckled +	do. —	...	8 do.
EC.	28-3-25	31-3-25	Hæmorrhagic ++ +	do. +	Congested —	do. —	...	3 do.
GC.	31-3-25	5-4-25	do. +	do. ++ +	do. +	do. +	...	5 do.
IC.	5-4-25	15-4-25	Caseous +	do. +	do. —	do. —	...	10 do.
LO.	15-4-25	3-5-25	Congested —	Nil —	Nil —	Nil —	...	18 do.
PC.	3-5-25	15-5-25	No signs	of plague.	12 do.

Series G. to PC. contain the greatest number of acute toxæmic cases.

The two series A. to DB. and ZC. to LD. include the largest number of the more advanced pyæmic cases.

It is a curious fact that in no instance were the kidneys observed to be the site of abscess formation macroscopically.

Four guinea-pigs showed a definite broncho-pneumonia but in many others there was an acutely congested condition of the lungs. In two cases there was an acute inflammatory condition of the bronchial glands although there was no definite consolidation of the lungs. Smears from these glands showed innumerable *B. pestis*. In two cases also there was a fibrinous pleurisy, without obvious hepatisation of the lungs.

Pericarditis with effusion is noted in five animals.

Whilst the experiments have shown that the bacillus recovered from human pneumonic plague is capable of causing bubonic and septicæmic plague in guinea-pigs, they have also shown that a broncho-pneumonic condition can be produced in a guinea-pig, with the organism obtained from a human case of bubonic plague. In these instances, however, the infection was a general one, *i.e.* implicating the lymph-glands, the liver and the spleen.

THE RODENTS OF LAGOS AND THEIR ECTOPARASITES.

The "rat campaign" initiated in August, 1924, has been prosecuted vigorously throughout the present year. In the previous report, details were given concerning 13,147 rodents. The total for 1925 is 151,007, giving a grand total 167,154 in eighteen months.

The identification and dissection have been in the hands of Mr. E. F. Hines and Corporal Bowrey, R.A.M.C., the former working until he proceeded on leave in April, and the latter from then to the end of the year. Most of the Ebute Metta, Iddo and Agege rodents, however, were examined here in Yaba.

The animals dealt with are *Rattus rattus*, *R. norvegicus*, *Mus musculus*, *Cricetomys gambianus*, *Crocidura manni* and *Lemniscomys fasciatus*. For the original identification of the latter two, thanks are due to Mr. Hinton of the British Museum.

Amongst the black rats there were many more or less marked variations in colour, and, to a less extent, in build. Some came near to *R. rattus frugivorus*. With the Agege rats particularly there was evidence of inter-breeding with different species. The scarcity of *C. gambianus* is remarkable as compared with the frequency of its occurrence at Accra, on the Gold Coast.

It will be seen from Table (5) that mice greatly outnumbered all the others. In fact, so numerous were they that from June onwards only a small proportion of them were dissected. Therefore, the number of plague-infected rodents is probably understated.

For the detection of plague-stricken animals, a smear from the spleen was taken. It was found impossible to examine the inguinal, axillary and cervical glands with the small staff available.

Carbol thionin blue was the principal stain used, as by it the bipolarity of the bacillus was best demonstrated. It may be of interest to record, in this connection that in a positive smear the violet tint of the stain is altered to a slate-blue, in practically every instance. It can be said, with some degree of accuracy that if all the slate-blue smears are taken they will be found to contain all the "positives." Some of these, of course, will show organisms other than *B. pestis*. A normal spleen smear has never been observed to give this changed colour.

TABLE (5).

Month.	Rattus. rattus.			Rattus norvegicus.			Mus musculus.			Crocidura mami.			Lemniscomys fasciatus.			Cricetomys gambianus.			Monthly Total.	Plague infected.			Total.
	Lagos.	Ebute-Metta.	Iddo.	Lagos.	Ebute-Metta.	Iddo.	Lagos.	Ebute-Metta.	Iddo.	Lagos.	Ebute-Metta.	Iddo.	Lagos.	Ebute-Metta.	Iddo.	Lagos.	Ebute-Metta.	Iddo.		R. rattus.	R. norvegicus.	M. musculus.	
January ...	1,042	350	115	55	2	2	3,816	714	35	220	57	76	...	2	1	6,487	7	...	5	12
	1,507			59			4,565			353			3			...							
February	974	290	99	96	4,547	915	33	243	53	35	...	2	1	7,288	1	...	2	3
	1,363			96			5,495			331			3			...							
March ...	999	286	131	203	...	1	4,942	1,001	43	181	42	20	...	1	2	7,852	1	1
	1,416			204			5,986			243			3			...							
April ...	1,041	294	120	95	4,206	1,195	121	158	31	10	...	1	1	...	7,273
	1,455			95			5,522			199			1			...							
May ...	1,387	343	111	186	...	1	6,199	1,337	210	177	22	5	1	...	9,979	1	1
	1,841			187			7,746			204									
June ...	2,837	420	106	320	9,986	967	179	261	22	15,098	9	9
	3,363			320			11,132			283									
July ...	3,405	452	82	456	10,956	1,164	174	252	17	1	16,959	8	2
	3,939			456			12,294			270									
August ...	3,224	386	231	473	1	...	10,385	1,139	120	203	23	7	16,192	5	1	...	6
	3,841			474			11,644			233									
September	2,822	382	80	535	2	...	10,342	1,036	143	131	14	2	15,489	35	5	...	40
	3,284			537			11,521			147									
October ...	3,558	306	115	454	...	1	10,957	997	110	174	4	2	16,678	65	8	...	73
	3,979			455			12,064			180									
November	3,208	188	110	461	5	...	10,897	791	94	120	4	12	15,980	52	6	...	58
	3,596			466			11,782			136									
December	2,708	129	134	442	10	2	12,861	615	162	118	6	6	17,193	65	6	...	71
	2,971			454			13,638			130									
Total for Year...	27,297	3,826	1,434	3,776	20	7	100,094	11,871	1424	2,238	295	176	...	6	4	...	2	...	152,468	247	26	9	282
	32,555			3,803			113,389			2,709			10			2							

TABLE (6).—AGEGE.

Month.	R. rattus.	M. musculus.	C. manni.	L. fasciatus.	Total.	Plague infected.
March ...	34	11	1	...	46	5 rattus 1 musculus.
April ...	76	32	1	...	109	1 rattus.
May ...	52	27	2	...	81	...
June ...	141	68	...	3	212	...
July ...	182	83	2	4	271	...
August ...	79	14	4	1	98	...
September ...	16	17	1	...	34	...
October ...	25	11	36	...
Total ...	605	263	11	8	887	6 rattus 1 musculus.

TABLE. (7).—APAPA.

Month.	R. rattus.	R. norvegicus.	M. musculus.	C. manni	Total.
August ...	49	4	...	12	65
September ...	109	7	21	4	141
October ...	64	5	106	17	192
November ...	40	3	88	4	135
December ...	41	6	65	7	119
Total ...	303	25	280	44	652

Rattus rattus outnumbered *R. norvegicus* in the proportion of about nine to one. *Crocidura manni* occurred only slightly less frequently than *R. norvegicus*. The other two types of rodent, *L. fasciatus* and *C. gambianus*, were comparatively rare.

Plague-infected rodents were found each month of the year, with the exception of April. In March and May however, the total was only one. October gave the highest number of positive findings (73), and the latter half of the year accounted for 256 in the total of 282 positives. There were many more infected black than infected brown rats (247 to 26), but proportionately there were nine of the former to seven of the latter.

No infected rodents were found either in Ebute Metta or Iddo. The records, indeed, showed that two occurred in Ebute Metta and one in Iddo but this is due in all probability to error on the part of the rat-catchers. As already stated, only a certain number of mice were dissected in the latter half of the year, so that the percentage of these infected cannot be given.

In March, a small outbreak of plague occurred at Agege, a village 15 miles directly inland by rail from Lagos. The rodents obtained showed six black rats and one mouse infected, for March and April.

No specimens were received after October so that, as Table (6) shows, 887 animals were identified and dissected in eight months. The Table gives the details.

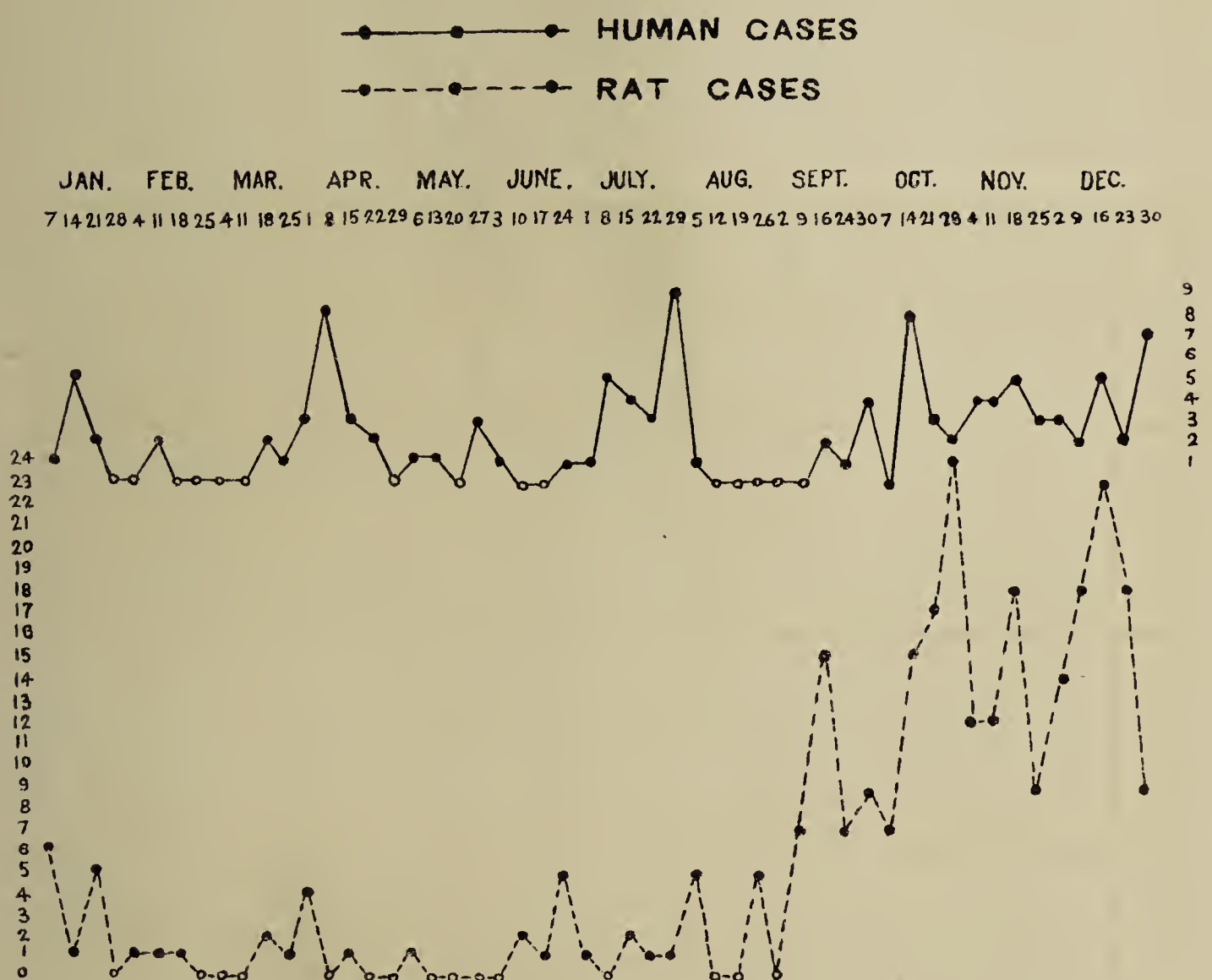
Collecting began at Apapa in August. Table (7) gives the figures from then until the end of the year. There were no positive findings.

TABLE (8).

Month	XENOPSYLLA CHIEOPIS.				X. BRASILIENSIS.				LÆLAPS ECHIDNINUS.			Total.		
	Lagos.		Ebute Metta.		Lagos.		Ebute Metta.		Agege.	Lagos.	Ebute Metta.		Agege.	
	♂	♀	♂	♀	♂	♀	♂	♀						
January	6	4	12	16	♂	♀	...	14	10	...	79	
February	13	11	...	3	16	...	55	
March	4	17	26	2	2	3	3	72	
April	15	17	2	7	1	...	3	10	69	
May	21	11	...	7	19	...	70	
June	18	23	...	12	5	2	66	
July	9	26	...	3	31	1	82	
August	1	1	...	1	5	...	11	
September	7	25	...	13	4	...	57	
October	3	1	3	5	...	1	...	3	8	...	37	
November	
December	
			18		277		15	13	137	1	17	104	16	598

CHART SHOWING ACTUAL NUMBERS OF HUMAN AND OF RAT PLAGUE IN LAGOS

DURING 1925.



ECTOPARASITES.

The ectoparasites of rodents identified during 1925 correspond with those given in the 1924 Annual Report. They are *Xenopsylla cheopis*, *X. brasiliensis* and *Laelaps echidninus*. The method of collecting these insects was the same as that previously employed, namely, filtering the disinfectant fluid, in which the dead rats were brought to the Institute, through a sieve and washing out the retained matter into a white enamelled basin. For various reasons this procedure was not followed out in Lagos, where most of the rats were dissected, so that the figures in Table (8) refer mainly to the combined districts of Ebute Metta and Iddo up till October.

The number of live rats obtained was small. In March two were received in a single cage. They yielded *X. cheopis* 4 ♂ ♂. Three rats were brought alive in September. One had seventeen fleas, one had four fleas and a *Laelaps* and one had none. They were identified as *X. cheopis* 3 ♀ ♀, 10 ♂ ♂ *X. brasiliensis* 4 ♀ ♀ 4 ♂ ♂ and *Laelaps echidninus* one. The foregoing rats were all *R. rattus*. In October, three *R. norvegicus* were received in one cage, also one single *R. rattus*, and two *R. rattus* in one cage. The first three carried *X. brasiliensis* 1 ♀ 5 ♂ ♂, the single rat *X. cheopis* 1 ♀ 1 ♂ and the last two rats *X. cheopis* 2 ♀ ♀ and *Laelaps echidninus* three.

Some interesting experiments similar to those done in India were carried out. On 8th January, two guinea-pigs were allowed to run loose in a room from which a plague corpse had been removed a few hours earlier. Twenty-one fleas were received from these animals, *X. brasiliensis* nineteen, and *X. cheopis*, two. Of this number nineteen insects had fed on the guinea-pigs within half an hour. In twelve of them only fresh blood was found and in the remaining seven there was some old blood as well. All were negative for *B. pestis*.

On 17th January the experiment was repeated in a house which had been disinfected twenty-four hours previously. The walls of this house, however, on two sides consisted of a double row of palm mid-ribs, between which the guinea-pigs disappeared. On recovering them twenty minutes later, they were found to have thirty-three fleas, *X. brasiliensis* twenty-eight and *X. cheopis* five. Twenty-eight had blood in the stomach, fresh only in nine, old and fresh in six and only old blood in thirteen.

Smears showed no *B. pestis*.

On the same day and in the same area, two other guinea-pigs were allowed to run loose for two minutes in a non-infected house. Several large cracks and holes were stuffed up to prevent the guinea-pigs escaping. No fleas were obtained.

On 19th January, two guinea-pigs were allowed loose for two minutes in a plague house which had been disinfected forty-eight hours previously. The walls and floor showed holes and large cracks. Two fleas were recovered, both *X. cheopis*.

Finally, on 12th February, two guinea-pigs were allowed freedom in a plague room which had been disinfected on the previous day by spraying with kerosene and cyllin. No fleas were obtained. These experiments indicate the inefficiency of gaseous disinfection of the average Lagos native house and point to the efficacy of thorough spraying with an oily compound.

It remains to express thanks to Mr. Jordan for the original identification of the fleas, and to Dr. Guy A. K. Marshall, C.M.G., F.R.S., for the original identification of the *Laelaps*.

YELLOW FEVER.

Yellow fever was unusually severe in Nigeria and particularly in Lagos. As is usual, when this disease makes its advent, the source of the infection is difficult to trace, and, on looking back one or more cases appear to be suspicious. Such cases, however, although they are borne in mind, are not included in the present report, because the speculative aspect exceeds the probable. For convenience and brevity, the clinical features, the gross post-mortem appearances and the histological findings are recorded in a table (Table (9)).

In addition, a certain number of cases occurred, which were probably more or less mild yellow fever, ending in recovery.

As regards the fatal cases there were fifteen in all. Seven occurred within the Lagos township, four in Ibadan, two in Warri and two in Port Harcourt. All the Lagos cases were in Europeans, one of the victims being a Greek and the others British. Of the four in Ibadan, one was a Syrian, another an African and two were British. Both the Warri cases were British as was one of the Port Harcourt cases. The other Port Harcourt case was a native African. The clinical histories have been supplied by Dr. G. M. Gray, Dr. A. B. Aitken, Dr. O'Keeffe, Dr. Morehead, Dr. Leonard, D.S.O., and Dr. Parkinson, with the kind collaboration of Dr. H. Beeuwkes, Director of the West African Yellow Fever Commission (International Health Board, Rockefeller Foundation).

Blood smears, taken in life, were examined from all the cases. In the two from Warri, the Medical Officer in charge demonstrated subtertian malarial parasites. In none of the others was a parasite seen. Further investigation was done, during life, in the Lagos cases, but this aspect will be considered later.

All the sections were made from tissues preserved in Formalin and stained with Hæmatoxylin and Eosin. In every case, also, pieces of tissue were stained by the Levaditi method. In some of the cases, other methods were used such as freezing and staining by Sudan III or Scharlach R. for fat, van Gieson for fibrous tissue, Pyronin Methyl green for plasma cells, and Giemsa for spirochaetes. Dried smears from organs were also examined after staining by Fontana or Giemsa, and fresh smears were examined under dark ground illumination for *Leptospira*. Dark ground work was uniformly negative as were also the stained smears. In one case only, that of H.L. of Lagos, in a single specimen of section of the liver, two bodies were found in a Levaditi preparation, very like *Leptospira*, but other sections of the same liver failed to show them.

An attempt to isolate *Leptospira icteroides* during life was unsuccessful as the following statements show. The reasons for this failure may be technical, or they may be due to the possibility that the cause of West African Yellow Fever is a more or less different organism than that causing the disease in America.

Case W.N. (fatal) sixth day of illness. Ten cc. blood from vein mixed with citrate and triple centrifuged. Final sediment examined under dark ground, and after fixation and staining by Giemsa and by Fontana, no *Leptospira* seen. 5cc. blood from a vein injected directly into the peritoneal cavity of each of two guinea-pigs. Animals showed no sign of illness.

Case M.N. (fatal) second day of illness. Same procedure, same result.

Case G.R. Recovered. Second day, same procedure, same result.

Case R.T. (fatal) third day, same procedure, same result.

Case M.L. Recovered. Second day, as before.

Case R.G. (fatal) Third day. guinea-pigs as before, neither showing signs of yellow fever.

Amounts of blood from 0.2cc. to 2cc. put into suitable tubes of locally prepared Noguchi medium and also into Wenyon's medium, twenty-two tubes in all. Result, no *Leptospira* seen, within one week. A few tubes contaminated.

Case K.T. Recovered. Third day. Guinea-pigs inoculated, as described and remained healthy.

Amounts of blood varying from 0.5 to 3cc. into six tubes Noguchi medium and six tubes Wenyon's medium. Results negative.

Case N.G. Recovered. Third day. Same procedure, same results.

„ C.R. „ Second day „ „ „ „

Blood smears taken on the day of admission to Hospital and others on succeeding days were examined, from all the cases. The malaria parasite was not seen in any. Differential leucocyte counts were done in most cases.

The figures of these are:—

Case.	Day of illness.	Poly.	Sm. lymph.	L. lymph.	Mono.	Eos.	Trans.	Mycho.	Mast.
G.R.	1st	74.2	5	1	15.4	0.6	3.6	0.2	—
M.N.	2nd	75.6	5.8	2.2	10.8	1.4	3.8	—	0.4
R.T.	3rd	81.6	9.6	1.6	5.6	0.4	1.2	—	—
S.L.	6th	40.8	13.6	3.2	28	10	4.4	—	—
R.G.	2nd	71.2	9.2	2	16.8	—	0.8	—	—
K.T.	2nd	73.2	6.8	2	15.6	0.8	1.6	—	—
N.G.	2nd	53.6	19.2	7.2	17.6	—	2.4	—	—
C.R.	2nd	70.8	7.6	2	17.6	—	2	—	—

The above counts are very similar except in Case S.L. a Syrian woman and Case N.G. a doubtful diagnosis. Examination of the urine revealed granular casts in all the cases. Dark ground examination of the sediment after rapid centrifugation, failed to reveal spirochaetes. Details of four feeding experiments with *Aedes argenteus* on four of the cases, are given under the heading "Entomological."

It will be noted that the epidemic was most severe in Lagos during April and May. During July, a few suspicious cases occurred. They all recovered and they were all thoroughly investigated by the West African Yellow Fever Commission.

BLACKWATER FEVER.

Reports of twenty cases have been furnished and they form the basis of the present analysis.

Nationality. British seventeen, Syrian one, West Indian one, Indian one.

Occupation. Officials eleven, non-officials nine. In the last group, in three cases patient was the wife of an official. Of the officials, six belonged to the Railway, two Engine Drivers, a Foreman-Platelayer, and Drill Foreman, a Workshop Artisan, and a Clerk. There were two in the Public Works Department, an Inspector and a Foreman. There were two also in the Marine Department, an Engineer and a Beach-master. The last was an Inspector in the Education Department.

Sex. Male seventeen, female three.

Age. There were five cases in the age-period 20–29 (22, 22, 25, 26, 28), four in period 30–39 (32, 32, 34, 34) and ten in period 40–49 (40, 40, 40, 42, 42, 44, 44, 45, 48, 49). The age of one patient is not stated.

Time of Year. January three, March two, May one, June two, July two, August four, September two, November one, and December three.

Locality.—The Southern Provinces provided eleven cases and the Northern Provinces supplied nine.

The actual stations are Kaduna two cases, Lagos two cases, Port Harcourt two cases and one case at each of the following, Aba, Afikpo Road, Badagry, Benin, Brass, Degema, Ebute Metta, Kano, Jos, Lokoja, Minna, Offa, Yola and Zaria.

Mortality.—There was a fatal issue in three cases, in all of which the patient was aged 40 years or over. The illness was an acute one in each instance death occurring within three days of the onset of hæmoglobinuria.

Previous Tropical Experience.—This is noted in fifteen cases.

Case (1) East Africa two years. Case (2) Singapore two years. Case (3) India and Mesopotamia three years. Case (4) Costa Rica three years, West Africa three and half years. Case (5) Nigeria ten years. Case (7) Nigeria two and half years. Case (12) Nigeria one year. Case (13) "Tropics" twenty-five years including five years in Nigeria. Case (14) "Some years." Case (16) Dahomey two and half years, Nigeria two and half years. Case (18) Nigeria two years. Case (19) India and Kenya fourteen years. Case (20) Nigeria six years.

Length of present Tour.—This is noted in nine cases.

Case (4) five months. Case (5) five months. Case (7) six months. Case (12) twelve months. Case (13) eight months. Case (14) three years. Case (16) five years. Case (18) nine months. Case (20) thirteen months.

Quinine Prophylaxis.—Case (1) took a five grain tabloid about once a week. Case (2) fifteen grains in tabloid form daily for ten months. Case (3) irregular. Case (4) five grains Bisulphate thrice weekly or more seldom. Case (5) two and half grains daily. Case (6) five grains Hydrochloride daily. Case (7) five grains daily. Case (8) took no quinine. Cases (9) and (10) irregular. Case (11) five grains daily. Case (12) Sometimes missed the daily dose. Case (13) irregular. Case (14) non-taker. Case (15) non-taker. Case (16) took quinine only when fever present. Case (17) five grains regularly. Cases (18) and (19) irregular. Case (20) misses quinine at times.

Previous Malarial attacks.—These vary from "none for seven years" Case (5), through "slight attacks" to "frequent attacks" except in Case (1) who stated he has had no malaria, and in Case (20) who stated he had no attacks recently.

The association of the subtertian malarial parasite with the actual condition of blackwater fever is demonstrated in ten cases.

Case 1.—Subtertian rings on third day. Quinine given in increasing doses appeared to hasten recovery.

Case 3.—Subtertian rings on second day. Improved under quinine.

Case 4.—Subtertian rings three days before onset of hæmoglobinuria.

Case 6.—Pigmented mononuclears on second day.

Case 8 is of considerable interest. She arrived in Nigeria in December, 1924. The patient took no quinine and for six weeks before coming under observation on 18th June, 1925. There had been repeated attacks of malaria for which no quinine had been given. On admission to Hospital on 18th June, the patient was nearly comatose, in a condition of extreme exhaustion. The Medical Officer in charge pointed out to the husband that if patient's life was to be saved, quinine must be given, but that in all probability blackwater fever would ensue. The drug was given in one-grain doses on 18th June, thrice in the day. The dose was increased to two grains and given four times on June 19th. On June 20th, three grains were given three times, similarly on 21st

TABLE (9).

Case.	Clinical History.	Post-mortem findings.	Liver.	Spleen.	Kidney.	Other organs.
G.B. A.	Illness began evening of 22.4.25 with headache and vomiting and fever. 24.4.25. Fever. Drowsy. Hiccough. Convulsive seizure. Coma. Death early morning 25.4.25.	Slight general icterus. Liver enlarged, yellow. Kidneys, congested. Spleen, congested. Stomach, no erosions.	Marked round-cell infiltration of Glisson's capsule, with some proliferation of bile ducts. Diffuse fatty degeneration of liver cells. Capsule thickened, with islands of lymphocytes lying directly under it.	General congestion, some polymorphic and lymphocytic infiltration. Endothelial cells of sinususes swollen.	A sub-acute condition. Tubular epithelium low, and tubules widely dilated. Granular contents with cast formation. Slight lymphocytic infiltration of stroma and glomeruli. Capsular thickening.	
W.N. L.	Illness began, evening of 22.4.25, with fever, headache and vomiting. On 27.4.25 Epigastric discomfort. On 28.4.25 Icterus and albuminuria. On 1.5.25 Epistaxis, Hiccough, Petechiæ on neck. Death 3.5.25.	General lemon-yellow tint. Petechiæ on forehead and chest. Submucous hæmorrhages in stomach. Liver enlarged and yellow. Kidneys acutely congested. Spleen congested.	Round-cell infiltration of Glisson's capsule. Extensive degeneration and necrosis of hepatic cells, most marked centrally. Capsular thickening and sub-capsular infiltration.	Considerable congestion, with swelling of the endothelial cells. Capsular thickening.	Tubular epithelium high in type and apparently well-preserved.	
M.N. L.	Illness began evening of 29.4.25, with rigor, headache and fever. On 2.5.25 Vomiting. On 3.5.25 Albuminuria, slight icterus. On 4.5.25 Definite icterus. Epigastric discomfort, Hæmatemesis. Hepatic enlargement. 5.5.25 Death.	General lemon-yellow tint. Petechiæ, chest and abdomen. Hæmorrhagic erosions, stomach. Liver enlarged, yellowish. Spleen congested. Kidneys inflamed.	Infiltration around Glisson's capsule, mainly lymphocytic but with some polymorphs. Proliferation of bile ducts. Complete central degeneration of hepatic cells with hæmorrhages. Slight capsular thickening.	Congestion. The normal appearance is considerably distorted by the lymphocytic infiltration and the endothelial hypertrophy.	Very high tubular epithelium. Lumina almost closed. Slightly granular contents. Cell protoplasm granular and vacuolated but nuclear staining good.	
R.T. L.	Pyrexia evening of 2.5.25. 3.5.25 Suffusion of eyes, Headache, albuminuria. 5.5.25 Slight icterus. Slow pulse. Hiccough. Vomiting of altered blood. 6.5.25 Petechiæ head and neck. 7.5.25, Death.	Faint yellow tinge. Petechiæ chest and abdomen. Hæmorrhagic erosions, stomach and duodenum. Liver enlarged, yellow. Spleen congested. Kidneys congested.	Impossible to make out normal structure clearly owing to the diffuse hæmorrhages and necrosis of liver cells. Capillaries distended, hypertrophy of the endothelial cells. Lymphocytic infiltration around Glisson's capsule. Some regenerative attempts on part of liver cells.	Congested and lymphocytic infiltration.	Epithelium rather high in type. Lumina open. Colloid or granular contents. Nucleation good. Congestion of glomeruli marked, with hæmorrhages.	
S.L. I.	Illness began 25.4.25. On 30.4.25, bleeding gums, delirium, fever. Icterus. Hæmaturia. 3.5.25 Suppression of urine, Black vomit. Melæna, coma. Death in evening.	Liver enlarged and yellow. Erythrosis, stomach. Spleen slightly enlarged. Kidneys congested.	Portal tracts picked out by the round-cell infiltration. Lobulation obliterated by hæmorrhagic areas and fatty degeneration of liver cells. The nuclei of the intact cells vary greatly in size and show various mitotic changes.	Hæmorrhages. Infiltration by lymphocytes, and hypertrophy of endothelial cells.	Low epithelium. Nucleation fair. Granular and colloid contents.	
R.G. L.	Illness began evening of 16.5.25 with Headache, vomiting and fever. 16.5.25, Albuminuria. 18.5.25, Bleeding from gums. Dark vomit. Conjunctivæ icteric. 20.5.25, Icterus general. Melæna. Died.	General orange yellow tint. Hæmorrhagic erosions in stomach. Liver enlarged, yellow. Spleen enlarged, congested. Kidneys acutely congested.	Mixed polymorphic and lymphocytic infiltration of Glisson's capsule. Diffuse degenerative changes in liver cells. Capillary congestion. Cell nuclei show variation in size.	Intense congestion, the Malpighian bodies appearing as cell islands surrounded by masses of red blood cells. Endothelial hypertrophy and mixed polymorphic and lymphocytic infiltration.	Epithelium ragged and lumina contain granular material with here and there, hæmorrhages. Intense vascular congestion. Patchy infiltrative areas composed of polymorphs and lymphocytes.	

TABLE (9)—continued.

Case.	Clinical History.	Post-mortem Findings.	Liver.	Spleen.	Kidney.	Other Organs.
B.L. P.H.	Not yet available.	Not yet available.	Marked infiltration of intact lobules, with their component cells separated by distended capillaries and exuded cells, there an area of lobules whose cells are almost entirely replaced by vacuoles (fatty). A small zone of intact cells remains around each tract. No increase of fibrous tissue.	Congestion and hæmorrhage with marked dilatation of sinuses. The organ in places resembles lung tissue. Endothelial hypertrophy.	Epithelium high in type and ragged. Granular protoplasm. Vascular congestion.	
H.L. L.	28/7/25, Fever, malaise, headache. 31/7/25, Nausea. Headache. Sclera icteric. Albuminuria. Melæna. Hæmatemesis. Nausea. Hiccough. Epigastric tenderness. Death.	General lemon yellow colour. Stomach and duodenum ecchymotic. Liver, enlarged, boxwood spleen congested. Kidneys congested.	Tracts sharply defined owing to halos of round cells with some polymorphs. A zonal appearance, relatively healthy cells with large nuclei around the tracts. Near central vein the cells become more and more degenerated (fatty), many of them being foamy owing to fine fatty droplets. Centrally the liver cells are entirely replaced by hæmorrhages and necrotic tissue. Round cells and polymorphs form small groups in the subcapsular region. Distended bile ducts visible. Many hepatic cells show a stippled appearance owing to bile pigment as do kupfer cells also. Some attempt at regeneration is present.	Marked congestion. Some fibrosis. Hypertrophy of endothelium, some of these cells assuming a cubical shape. Numerous plasma cells present.	Low epithelium. Tubules separated by a delicate oedematous granulation tissue. Tubular contents mainly granular, with some colloid casts, showing evidence of calcification. Small hæmorrhages in the stroma. Congestion of vessels generally, including the glomerular capillaries.	Stomach. Congestion, superficial hæmorrhage and exudate forming a sort of membrane. Duodenum. Very marked congestion with hæmorrhage on surface. Pancreas. Endothelial hypertrophy. Lungs. Marked congestion and oedema, with diffuse hæmorrhages. In these organisms are present. Bronchi. Lining epithelium is replaced by a hæmorrhagic exudate. Many of the bronchi contain masses of organisms.
T.L. L.	8/9/25, Rigor and Fever. 10/9/25, Bilious vomit. Injected sclera. Epigastric tenderness. Black vomit. Albuminuria. 11/9/25, Delirium. Melæna.	General lemon yellow tint. Petechiae on face. Ecchymose in stomach. Liver, enlarged, congested. Kidneys, congested.	Infiltration of tracts. This liver does not show much differentiation into zones. The cells appear diffusely degenerated and necrotic. Around some of the portal tracts there are hepatic cells in a better state of preservation.	Marked congestion. Infiltration of Malpighian bodies.	High epithelium and granular material in many of the lumina. Acute vascular congestion including the glomerular capillaries.	Duodenum. Congestion and superficial necrosis of the mucous layer, with here and there membrane-formation of fibrinous material. Lung. Congestion and hæmorrhages into alveoli. Peribronchial infiltration with some desquamation of the lining epithelium. Pancreas. Well preserved. Brain. Congestion with diffuse round-cell infiltration.

TABLE (9)—continued.

Case.	Clinical History.	Post-mortem Findings.	Liver.	Spleen.	Kidney.	Other Organs.
P.R. W.	10/7/25 Rigors and headache, 11/7/25 Photophobia, nausea and bilious vomiting, Albuminuria, Fever, 12/7/25 Haematemesis, 13/7/25 Haematuria and epigastralgia. 13/7/25 Sclerae yellowish. Melena, 15/7/25 Bleeding gums, 16/7/25 Hiccough, Anuria, 17/7/25 Coma, Death.	General yellow tint. Echymoses, scrotum, stomach shows hæmorrhages, also small intestine, Liver enlarged, pale yellow boxwood. Spleen dark colour. Kidneys enlarged, congested.	Slight infiltration of Glisson's capsule, marked degeneration of hepatic cells, with hæmorrhages. Zone of well-preserved cells around Glisson's capsule.	Diffuse infiltration and fibrosis	Low epithelium, tubules granular contents. Glomerular congestion.	Small Intestine. Diffuse round cell infiltration of mucous membrane. Large Intestine. Similar condition with superficial hæmorrhage.
R.L. W.	15/7/25 Headache, 16/7/25 Fever, 17/7/25 Nausea and vomiting, 18/7/25 Injected conjunctival Epigastralgia, 19/7/25 Albuminuria, 20/7/25 Scleral jaundice, 21/7/25 Black vomiting. Melæna, Petechiæ face, thorax, abdomen, legs, Bleeding gums, General icterus, Coma, Death.	Skin yellow. Petechiæ general, stomach shows small patches of hæmorrhage. Duodenum small hæmorrhages, Liver yellowish brown, Spleen dark, Kidneys congested.	Lymphocytic infiltration of Glisson's capsule. Relatively well-preserved layer of cells around each portal tract, producing a net-like appearance, the meshes of which contain broken down liver cells, with cellular infiltration and hæmorrhage.	Infiltration of Malpighian bodies. Pulp congested and slightly fibrotic.	High epithelium, lumina mostly obliterated. Here and there, granular contents. The glomeruli completely fill up Bowman's capsule. Vascular congestion. No capsular thickening.	
V.N. P.H.	5/6/25 Fever, slow pulse. Albuminuria appeared to improve until 9/6/25 when mild delirium appeared, suffusion of eyes and epigastric tenderness, with slight jaundice. Later black vomit and Haematuria, death in evening.	Not yet available.	Infiltration of Glisson's capsule. Marked necrosis with hæmorrhages in centre of each lobule. The liver cells surrounding portal tracts are better preserved and show some attempt at regeneration.	Congestion of pulp and marked dilatation of sinuses. Diffuse fibrosis.	Epithelium low in type some glomerular congestion.	Pancreas apparently normal. Stomach. Nothing of note.
D.M. I.	Admitted 9/6/25 with fever and headache, died on 11/6/25 just after black vomit set in.	Liver enlarged, yellow. Stomach, ecchymotic. Spleen slightly enlarged, Kidneys congested.	Infiltration of portal tracts. Central degenerative changes.	Markedly congested.	High type of epithelium with granular material in lumina of tubules. Glomeruli congested and a granular oedematous exudate between tuft and capsule. Vascular congestion.	Small Intestine. Diffuse lymphocytic infiltration of submucosa and mucous coat.
P.R.N. I.	Illness began 15/8/25, with chill, headache, and body pains, 18/8/25 conjunctival suffusion, Nausea, Albuminuria, Haematemesis, 19/8/25 Sclerae yellow, Hiccough, vomiting, Epigastric tenderness, 20/8/25 Petechiæ, shoulder, 21/8/25 General icterus, 22/8/25 Delirium. Coma, Death.	Icterus general, Liver, yellowish brown. Stomach, hæmorrhagic areas, Spleen congested. Kidneys congested.	Infiltration of portal tracts. Marked zonal appearance in each lobule. Around each portal tract is a band of intact liver cells, then an area of fatty degenerated hepatic cells and in the centre a mass of extravasated red blood cells and necrotic liver cells.	Marked congestion with dilatation of sinuses.	Marked acute condition. Epithelium high, and granular. Glomeruli show marked capillary congestion. Infiltration and oedema of stroma tissue, mainly about the glomeruli. Component cells are mixed round and polymorphic. Capsule thickened and oedematous.	
T.R. I.	9/10/25 Headache, fever and vomiting, 10/10/25 Albuminuria, 12/10/25 Bloody vomit, 13/10/25 Jaundice, Melæna, Death.	Liver enlarged, yellow, Hæmorrhages, stomach, Spleen not enlarged. Kidneys congested.	Diffuse and almost complete necrosis of hepatic cells. Impossible to make out any structure apart from the tracts.	Congestion.	Moderately high granular epithelium. Lumina contain a granular material. Marked congestion.	

and 22nd June. On 23rd June five grains were given three times. On 24th June five grains were given at 11 a.m. and hæmoglobinuria was observed four and half hours later. The attack lasted just under two days, and was a mild one. The Medical Officer believes that with even more cautious dosage the hæmoglobinuric condition might have been avoided, for the reason that the destruction of parasites would have been more slow and therefore the liberation of toxic products proportionately delayed and lessened. This belief must appeal to those who have studied and treated cases of blackwater fever.

Case 11.—Subtertian rings appeared, on a rise of temperature on the day the urine cleared up, the hæmoglobinuria having set in three days previously.

Case 12.—Subtertian rings present on second day.

Case 16.—Subtertian rings on 1st day.

Case 18.—Subtertian rings on 1st day.

Case 20.—Pigmented mononuclears 1st day.

When it is considered that in many of the other cases the patient was not seen until late in the disease, these facts are the most striking yet obtained in the series of Annual Reports presented from this Colony.

QUININE ADMINISTRATION PRIOR TO ATTACK OF HÆMOGLOBINURIA.

Case 1, who took five grains of quinine about once a week, had that dose sixty hours before blackwater appeared.

Case 2 had been taking fifteen grains daily in a single dose for ten months previously. Four hours elapsed between the last dose of quinine and the onset of hæmoglobinuria.

Case 3 had been taking ten grains daily for “some days” before, his habit having been irregular previously. His last dose however, was over forty-eight hours before the onset of “blackwater.”

Case 4.—He took five grains Bisulphate perhaps thrice weekly. On 5th March he took ten grains in solution. On 6th March he took fifteen grains in solution. On 7th March in two doses of ten grains, one in the morning and the other in evening he took twenty grains. On 8th March he took ten grains in solution at 9 a.m. At 2.30 p.m. he noticed “black urine.”

Case 5.—For nine years had been taking two and half grains quinine in liquid form, daily. For a week previous to the appearance of blackwater he had been taking ten to twenty grains in ten grain doses daily. There was an interval of six hours between the last dose of quinine and the passing of blackwater.

Case 6.—Stated he took “five grains daily.” On 24th May, took five grains in tabloid form by mouth four times. Again on 25th took five grains in solution four times, last dose at 1 p.m., hæmoglobinuria observed two hours latter.

Case 7.—Took five grains Hydrochloride daily and on one occasion a week before the appearance of blackwater, took ten grains. There was an interval of four and half hours between the last five grains of quinine and the passing of typical urine.

Case 8.—The full history of this case, as regards quinine, has already been given.

Case 9.—“Sometimes took five grains daily.” Ten grains were taken at 6.30 a.m. 4th July. Blackwater was passed at 1.30 p.m. on the same day.

Case 10.—Took five grains quinine occasionally. Took ten grains Hydrochloride on 5th July, 1925 and next day passed blackwater.

Case 11, was accustomed to take five grains daily as a prophylactic. He had five grains Hydrochloride at 6 p.m. on 2nd August and sixteen hours later blackwater appeared.

Case 12.—From August 1924 until January 1925 took two and half grains daily. Thereafter took five grains daily Hydrochloride until evening of 16th August, 1925, when she took ten grains Hydrochloride. An interval of seven hours elapsed before the voiding of blackwater.

Case 13.—Took five grains Hydrochloride in tabloid form, but not regularly. On the evening before the appearance of blackwater he took five grains in solution, but vomited the dose immediately.

Case 14.—Took no quinine.

Case 15.—Took no quinine.

Case 16.—His usual custom was to take quinine only when he had fever. However, on the present occasion he had taken no quinine for three months, when after having had fever for two days, he developed blackwater.

Case 17.—Took five grains regularly. On 27th October he took fifteen grains in tabloid form. Thereafter he took five grains daily until 30th October. The last dose in the form of a tabloid, grains five, was taken at 7 p.m. Patient passed blackwater thirty-six hours later.

Case 18.—Took quinine Hydrochloride not very regularly. Two hours before appearance of blackwater, took fifteen grains Hydrochloride.

Case 19.—"Perhaps missed twice a week taking five grains daily in solution." Took five grains Hydrochloride on each of two mornings previous to onset of hæmoglobinuria. Interval between last dose and passing of blackwater twenty-four hours.

Case 20.—Did not take quinine regularly but on 28th December took the following, 3.30 a.m. five grain tabloid Hydrochloride, 2 p.m. two tabloids ditto, 7.30 p.m. five grains in solution; dark urine six hours later.

TABLE (10).

Case.	I. Quinine.	Interval.	P. Quinine.	Total Quinine 48 hours.
1	5 grains	60 hours	—	—
2	15 do.	4 do.	15 grains	30 grains
3	10 do.	48 do.	—	10 do.
4	10 do.	5½ do.	50 grains	30 do.
5	10-20 do.	6 do.	10-20 do.	20-40 do.
6	20 do.	2 do.	20 do.	40 do.
7	5 do.	4½ do.	5 do.	10 do.
8	5 do.	4½ do.	15 do.	20 do.
9	10 do.	7 do.	—	10 do.
10	10 do.	24 do.	—	10 do.
11	5 do.	16 do.	5 grains	10 do.
12	10 do.	7 do.	5 do.	15 do.
13	5 do. (vom- ited).	12 do.	nil.	—
14	none.	—	nil.	—
15	none.	—	nil.	—
16	none.	—	nil.	—
17	5 grains	36 hours	nil.	5 grains
18	15 do.	2 do.	nil.	15 do.
19	5 do.	24 do.	5 grains	10 do.
20	20 do.	6½ do.	nil.	20 do.

Table (10) puts the facts shortly. The column "I. Quinine" gives the dosage of quinine last administered. "Interval" refers to the time elapsed between this last dose and the voiding of black urine. "P. Quinine" is the amount taken in the twenty-four hours previous to the last dose. In cases (1), (13), (14), (15) and (16) the onset of hæmoglobinuria cannot be attributed to quinine.

A previous history of blackwater fever is given in three cases, Case (9) (West Indian) in 1922, Case (10) (Indian) in 1906 and 1921, and Case (11) twice previously.

A remission occurred in three cases. Case (2) showed alternately red and clear urine from the 2nd to the 5th day, Case (3) on the 5th day and Case (7) on the 3rd and 5th day.

A relapse occurred in three cases, Case (6) cleared on the 4th day, relapsed on the 5th and cleared on the 6th day, Case (15) cleared on the 2nd day, became red on the 3rd and cleared on the same evening, Case (16) cleared in two days, remained clear for eight days and was red again for one day.

The total duration of hæmoglobinuria in the cases which recovered was twelve hours in two cases, one day in two cases, two days in three cases, four days in one case, five days in two cases, six days in two cases and seven days in one case. There was no information in one case. In the three fatal cases, the hæmoglobinuria was present throughout the three days' illness of each.

As regards the signs and symptoms during the attack a perusal of the reports brings no unusual features to light.

A number of differential leucocyte counts and Arneth counts were made. These are given in Table (II).

LEPROSY.

In the previous year's report eight patients were under treatment by Moogrol intravenously. This was continued in each until the 1st of June by which time all were obtaining their maximum dose of 4 to 6cc. Early in January, a former inmate who had disappeared, returned and asked for treatment and in March a recent prisoner also asked "for the medicine". From June until October Moogrol was suspended and each patient was given Copper Sulphate by the mouth in half-grain doses daily. At the end of that period, seven patients had seriously retrogressed and three showed no improvement. Accordingly, a new line of treatment was adopted. On Monday of each week, Sodium Morrhuate was given subcutaneously and on Thursday of each week Ethyl Hydnocarpate by the same route.

The initial dose of Sodium Morrhuate was 0.5cc. of a 3% solution rising at each injection by 0.5cc. until an amount of 2.5cc. was reached. The initial dose and the increase were the same with the Hydnocarpate. When the Sodium Morrhuate had reached 2½cc., it was discontinued and Hydnocarpate was given twice weekly. All the patients have willingly undergone the treatment. During the year the prisoner was released and volunteered to return as an inmate. A female patient also asked for treatment so that the number of cases is now eleven. Some few signs of improvement are evident but the main consideration is that all the patients come forward for their twice weekly injections.

ENTOMOLOGICAL.

The most important investigation undertaken was an examination of the markings of *Aedes* (*Stegomyia*) *argenteus*, a much harassed insect both in nomenclature and in practice. One thousand males and one thousand females have been examined. They were not specially selected but were taken as they hatched out from the collections of larvæ sent to the Institute by the Medical Officer of Health.

TABLE (II).

Case.	Day of disease.	Parasites.	Pigment.	Polymorph.	Sm. lymph.	L. lymph.	Mono.	Eos.	Trans.	Mast.	Myelo.	Nucl. red.	Ery.	Vac.	I.	II.	III.	IV.
6	2nd	+	61·6	13·8	5	17·6	0·4	1·6	2	28	75·2	20	4·8	...
9	2nd	72·8	12	2	11·2	...	1·6	0·2	0·2	69·6	25·2	4·8	0·4
13	1st	58	9·6	2·2	26	...	3·4	0·8	62	27	9	2
14	1st	64·6	11·2	3·6	19·2	...	1·4	62·4	30·4	6·4	0·8
16	1st ...	+	+	78·4	6·8	1·6	11·6	0·4	1·2	88	10	2	...
	2nd ...	+	+	78	12	1·6	7·6	...	0·8	66	27	7	...
	3rd	81·2	13·6	2	7·2	0·4	67	24	8	1
	4th	71·6	8·4	3·2	15·6	...	0·8	...	0·4	58	29	13	...
	5th	70·4	8	1·6	12·4	2·4	3·2	0·4	1·6	2	69	26	5	...
	6th	73·6	10·4	2	9·2	...	3·2	...	1·6	2	69	26	4	1
	7th	76	8·8	3·2	9·6	...	1·2	...	1·2	3	64	29	6	1
18	1st ...	+	+	68·8	6	2	20·8	...	1·6	0·2	0·6	78·4	18	3·6	...
20	1st ...	-	+	64·2	9·8	2·2	18·6	0·6	0·4	...	4·2	1	...	5	64	26·4	8·8	0·8
	2nd	59·6	11·2	5·2	22	...	0·4	...	1·6	1	...	13	58	27	10	5

Ery=Erythrophages. Vac=Vacuolated Mononuclears. Roman figures refer to Arneth's classification.

Many micro-photographs were taken, to show variations in the marking of the abdomen.

The work of tabulation and sorting is still in progress so that a complete account cannot be given. It may be stated, however, that the characteristic lyre on the thorax is the feature least liable to variation as regards its shape. As regards the markings on the legs and the colour and the markings on the abdomen a very wide variation has been found to exist. A great many specimens have been obtained more worthy of varietal rank than any already given that status. The object of the work, however, is to lessen, not increase the number of varieties.

CROSS-BREEDING EXPERIMENTS.

In addition to a number of cross-breeding work with differently marked *Aedes argenteus*, the following two experiments were done:—

8th July. Two *Aedes luteocephalus* ♂ ♂ and many *Aedes argenteus* ♀ ♀ all newly hatched, let loose in a bell-jar, containing fresh water and a lump of sugar. The females each had two feeds of blood.

26th July. All insects dead. No eggs laid. On dissection of females no eggs found.

23rd July. One *Aedes longipalpis* ♂ and one *Aedes luteocephalus* ♀ put in tube. Both freshly hatched. Male fed on sugar, female on human blood.

25th July. Male dead.

26th July. Female again fed on human blood.

Two *Aedes longipalpis* ♂ ♂ put in tube.

30th July. All insects dead. No eggs laid and none found on dissection of *Aedes luteocephalus*.

An attempt to obtain the eggs of *Eretomopodites chrysogaster* failed, by the following method.

23rd May. *Eretomopodites chrysogaster* 2 ♀ ♀ 1 ♂ bred from larvæ, put into a bell-jar containing fresh water, various flowers, a dish of sugar-water and a banana.

24th May. Fresh flowers supplied.

25th May. Fresh banana given.

26th May. Fresh flowers.

27th May. „ „

28th May. „ „

29th May. Fresh water, flowers, banana and a piece of sugar given. The fresh supplies were kept up regularly.

6th June. One ♀ dead. No eggs. (Dissected).

18th June. The ♂ dead.

23rd June. Second ♀ dead. No eggs. (Dissected).

DISSECTIONS.

The following wild biting insects were dissected in a search for parasites.

Taeniorhynchus (*Mansonioides*) *africanus* 72 ♀ ♀.

Spirochaetes in stomach contents 2.

Oocysts, stomach wall ... 2.

amoebæ in stomach contents ... 1.

sporozoit-like structures, in salivary glands ... 1.

microfilariae in stomach contents 1.

negative ... 65.

Anopheles (*Pyretophorus*) *costalis* 32 ♀ ♀.

Oocysts, stomach wall ... 1.

negative ... 31.

Culiciomyia nebulosa 10 ♀ ♀.

Innumerable minute nematodes in stomach contents 1.

negative ... 9.

Culex thalassius 5 ♀ ♀.

Spirochaetes in stomach contents 1.

negative ... 4.

Glossina palpalis 5 ♀ ♀.

Trypanosomes (? *vivax*) in proboscis 1.

negative ... 4.

Fannia sp. 5 ♀ ♀. *E. histolytica* cysts in stomach 1.

negative ... 4.

Anopheles funestus 1 ♀, *A. maculipalpis* 1 ♀ *Taeniorhynchus annetti* 1 ♀. *Aedes irritans*, 1 ♀ all negative.

A supply of mosquitoes came from the yellow fever areas in Lagos and Apapa.

Culiciomyia nebulosa 58 ♀ ♀. One had crithidia in stomach contents.

Aedes argenteus 53 ♀ ♀. One had spirochaetes in stomach contents.

Culex thalassius 42 ♀ ♀. One showed spirochaetes, another crithidia in stomach contents.

Anopheles costalis 21 ♀ ♀, all negative.

Aedes irritans 9 ♀ ♀ „ „

Taeniorhynchus annetti 7 ♀ ♀, one had spirochaetes in stomach contents.

<i>Aedes nigricephalus</i> ...	5 ♀ ♀	} all negative.
<i>Culex fatigans</i> ...	5 ♀ ♀	
<i>Taeniorhynchus africanus</i> ...	3 ♀ ♀	
<i>Lutzia tigripes</i> ...	2 ♀ ♀	

FLEAS.

These have been described under the heading of "The Rodents of Lagos and their Ectoparasites."

It has to be added, however, that in the fleas collected from guinea pigs which were allowed to run loose in plague houses the following showed an encysted nematode in the abdomen:

Xenopsylla cheopis 3 ♀ ♀ 1 ♂.

X. brasiliensis 3 ♀ ♀ 2 ♂ ♂.

FEEDING EXPERIMENTS WITH *Aedes argenteus* IN CASES OF YELLOW FEVER.

In the first experiment three *Aedes argenteus* were fed on a yellow fever patient in the third day of his disease. One was killed on the 9th another on the 13th and the last on the 23rd day after the initial feed. All were preserved and embedded in paraffin. They await sectioning.

In the second experiment four *Aedes argenteus* were fed on a patient in the third day of the disease. One was killed on the 11th, another on the 13th, a third on the 15th and the last on the 25th day after the infective meal. They were all preserved and embedded in paraffin.

In the third experiment three *Aedes argenteus* were fed on a patient in the second day of the disease. Two were killed on the 5th day and the last on the 28th day. They also were embedded.

In the fourth experiment seven *Aedes argenteus* were fed on a patient in the third day of illness. Two were killed on the 6th day, one on the 21st, one on the 23rd, two on the 30th and the last on the 32nd day after the infective feed.

All the insects were bred from larvæ, and they were kept alive with blood meals from guinea-pigs. One guinea-pig was reserved for each batch. None of the four guinea-pigs developed any febrile disturbance.

In a fifth experiment three *Aedes argenteus* caught in the bedroom of the patient in the second experiment were kept alive by feeding on a guinea-pig. One was killed two days later.

On the 10th day both of the remaining mosquitoes had laid eggs in a dry test tube. On the 15th day each had again laid eggs. One was killed on the 16th day and the third survived sixty-six days. All were preserved for section.

The guinea-pig on which they fed remained healthy.

SPECIMENS SENT FOR IDENTIFICATION.

The Medical Officer of Health, Lagos, sent the following:—

<i>Aedes nigricephalus</i>	19 ♀ ♀	1 ♂
<i>Aedes irritans</i>	10 ♀ ♀	1 ♂
<i>Culiciomyia nebulosa</i>	2 ♀ ♀	1 ♂
<i>Culex insignis</i>	2 ♀ ♀	
<i>Aedes argenteus</i>	2 ♂ ♂	
<i>Anopheles costalis</i>	2 ♀ ♀	
<i>Aedes argenteopunctatus</i>	1 ♀	
<i>Culex decens</i>	1 ♂	

Also the larvæ of a Dermestid beetle, obtained from stockfish.

Dr. Dyce-Sharp sent from Sokoto.

<i>Taeniorhynchus africanus</i>	1 ♀	
<i>Anopheles costalis</i>	2 ♂ ♂	

Dr. Bauvallet sent from Kotonu

<i>Culiciomyia nebulosa</i>	12 ♀ ♀	37 ♂ ♂
<i>Anopheles costalis</i>	4 ♀ ♀	4 ♂ ♂
<i>Taeniorhynchus africanus</i>	3 ♀ ♀	
<i>Culex grahami</i>	3 ♀ ♀	
<i>Aedes argenteus</i>	1 ♀	
<i>Lutzia tigripes</i>	1 ♀	

LARVAE RECEIVED FROM MEDICAL OFFICER OF HEALTH.

Six thousand five hundred and twenty-seven collections were received. The identifications are set out in Table (12) under headings of month, receptacle and mosquito.

Larvæ of eighteen species were found, namely :—

<i>Aedes argenteus</i>	in 3,700 collections of larvæ.
<i>Culiciomyia nebulosa</i>	in 2,059 collections of larvæ.
<i>Anopheles costalis</i>	„ 443 „ „ „
<i>Culex fatigans</i>	„ 196 „ „ „
<i>Culex duttoni</i>	„ 86 „ „ „
<i>Aedes irritans</i>	„ 58 „ „ „
<i>Aedes luteocephalus</i>	„ 55 „ „ „
<i>Culex decens</i>	„ 46 „ „ „
<i>Culex insignis</i>	„ 13 „ „ „
<i>Uranotænia annulata</i>	„ 12 „ „ „
<i>Lutzia tigripes</i>	„ 7 „ „ „
<i>Culex decens</i> var <i>invidiosus</i>	in 5 collections of larvæ.
<i>Aedes apicoargenteus</i>	in 4 collections of larvæ.
<i>Culex thalassius</i>	in 3 collections of larvæ.
<i>Aedes apicoannulatus</i>	in 2 collections of larvæ.
<i>Aedes nigricephalus</i>	in 1 collection of larvæ.
<i>Eretmopodites chrysogaster</i>	in 1 collection of larvæ.
<i>Aedes africanus</i>	in 1 collection of larvæ.

The collections from “Marine boats” and “Barges” came from the Port Health Officer and the remainder from the Medical Officer of Health.

Aedes argenteus, as usual, predominated and the commonest receptacle for breeding is the familiar “household” pot, an earthenware vessel for holding water.

INVESTIGATION OF THE SKIN LESIONS PREVALENT IN LAGOS.

Dermatological histology, though one of the most fascinating and important branches of pathology, particularly in the tropics, has, up to the present, attracted few workers in West Africa. French observers, particularly in Senegambia and in Dahomey and Macfie in the Gold Coast have published results of investigations, but no systematic attempt has yet been made to investigate and classify the numerous skin lesions.

Too much credence has been generally placed in the belief that syphilis is the cause of the majority of conditions met with, and although it is claimed that a large percentage of native Africans give a positive Wassermann reaction, it should not be assumed that in every case the lesions present are of spirochaetal origin. A thorough investigation of the dermal condition, both bacteriological and histological, in the present state of our knowledge, is necessary for a correct diagnosis.

Similarly the term “craw craw” may be said to cover a multitude of irritant eruptions.

With the object of determining with some degree of accuracy, the nature and variety of skin lesions prevalent among the African inhabitants of Lagos, an examination of all patients presenting themselves at the African Hospital, was commenced in August.

At the outset, it is necessary to express gratitude to the Resident Medical Officers and to the Nursing Staff for their active and ungrudging efforts, both in producing cases and in assisting the examination.

Upwards of one hundred cases have, in these five months been investigated more or less fully. It is pleasant to record, that with few exceptions, the patients take a keen interest in their own particular case and readily submit to examination and treatment.

TABLE (12).

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
<i>Agbo Pots.</i> Culicomyia nebulosa ...	2	3	4	6	0	4	11	4	11	6	12	1	64
Aedes argenteus	1	...	1	...	2	4
Culex duttoni	1	...	1
Culex fatigans and Culicomyia nebulosa	1	1
Cul. neb., Aedes arg., and Culex decens	1	1
Culicomyia nebulosa and Aedes argenteus	1	1
<i>Barrel.</i> Aedes argenteus	1	6	3	18	60	33	19	8	11	12	5	176
Culicomyia nebulosa	1	1	...	2	6	13	10	9	5	15	4	66
Anopheles costalis	1	10	5	16
Culex fatigans	3	2	2	1	1	9
Culex duttoni	1	1	...	1	...	3
Aedes argenteus and Cul. fat.	1	1	2
Aedes argenteus and Anopheles costalis	1	1	...	1	3
Aedes argenteus and Cul. decens	1	...	1	...	1	...	3
Culicomyia nebulosa and Culex fatigans	1	1	1	3
Culex fatigans and Culex inv.	1	1
Culex decens	1	...	1	2	...	3
Aedes argenteus and Aed. lut.	1	1
<i>Bottle.</i> Aedes argenteus	3	6	5	3	2	4	2	3	28
Lutz. tigr.	1	1
Cul. dec.	1	1
Cul. neb.	1	...	1	5	3	2	12
An. cost.	1	1
Aed. lut.	1	1
Cul. dutt.	1	1

TABLE (12)—continued.

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
<i>Bucket.</i> Aed. arg. Cul. fat. Cul. neb. An. cost. Aed. arg. and Cul. fat.	...	1	2	...	4	5	4	...	1	7	1	2	27
	1	1
	1	3	1	2	3	3	2	15
	1	1	2
	1	1
<i>Calabash.</i> Cul. neb. Aed. arg.	1	2	2	3	...	1	1	...	10
	1	4	...	2	...	1	1	1	10
<i>Canoe.</i> Cul. thal. Aed. arg. Cul. thal. and An. cost. Cul. neb. An. cost.	...	1	1
	1	1
	1	1
	1	1
	1
	1	1
<i>Car.</i> Aed. arg.	2	2
<i>Casting.</i> Aed. arg. An. cost.	1	1
	1	1
<i>Catchpit.</i> Lutz. tigr. and Cul. neb. Cul. neb. Cul. neb. and An. cost. Cul. fat. and Aed. arg. Aed. arg. Cul. fat. An. cost. Cul. dec. Cul. dutt. An. cost. and Cul. fat. Aed. lut. Aed. arg. and Cul. neb.	1	1
	1	...	1	1	2	3	13	17	30	27	43	25	163
	1	1
	...	1	1	1	4	7
	1	...	4	16	13	7	10	14	6	11	82
	3	...	1	4	9	3	6	4	1	...	31
	1	...	2	1	4
	2	...	1	1	1	...	5
	2	...	1	3
	1	1
	1	2
	1	4	1	...	6
	5
<i>Cooler.</i> Cul. neb. Aed. arg. Cul. dec.	1	1	1	2	...	5
	1	2	1	...	2	1	...	1	8
	1	1

TABLE (12) —continued.

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
<i>Crab-holes.</i> Aed. irr.	1	30	25	56
Aed. arg.	1	1
Cul. fat.	1	1
An. cost.	1	1
Cul. insign.	7	5	12
Ur. ann.	6	5	11
Aed. nigr.	1	...	1
Aed. irr. and Ur. ann.	1	...	1
Aed. irr. and Cul. insign.	1	1
Cul. fat. and Cul. neb.	1	...	1
<i>Drain.</i> Aed. arg.	2	3	4	...	1	5	3	...	18
Cul. fat.	3	3	1	1	8
Cul. neb.	1	1	1	1	6	3	9	22
An cost.	1	2	1	...	1	...	3
An. cost. and Cul. fat.	1	5	...	1	9
Cul. dec.	1	1
<i>Drum.</i> Cul. neb.	1	2	...	4	5	5	4	5	10	10	12	58
Aed. arg.	1	16	25	15	5	13	20	7	6	108
An. cost.	7	2	...	9
Cul. fat.	1	1	...	1	...	3
Culex duttoni	1	1	2
<i>Dye-pot.</i> Aedes argenteus ...	1	1
Cul. neb.	1	2	2	5
<i>Filter.</i> Cul. neb.	1	1
<i>Flower-pot.</i> Cul. neb.	1	1
Aed. arg.	6	2	2	1	11
Cul. fat.	1	1	2
Aed. arg. and Cul. fat.	1	1
<i>Go-cart.</i> An. cost.	1	1
Cul. neb.	1	1

TABLE (12) --continued.

	January	February	March	April	May	June	July	August	September	October	November	December	Total
<i>Grind-stone.</i> Aed. arg.	1	...	1	2
Cul. neb.	1	1
<i>Gutters.</i> Aed. arg.	1	1	2
<i>Ice-chest.</i> Aed. arg.	1	1	1	3
<i>Jug.</i> Aed. arg.	1	2	1	...	4	4	12
An. cost.	1	1	2
Cul. neb.	1	2	2	3	8
<i>Kettle.</i> Aed. arg.	2	4	1	...	2	1	1	2	13
Cul. dec.	1	1
Cul. neb.	1	3	4
Cul. fat. and An. cost.	1	1
<i>Life-boat.</i> Aed. arg.	1	1
<i>Mortar.</i> Cul. neb.	2	2
<i>Pail.</i> Aed. arg.	1	1
<i>Pan.</i> Aed. arg.	6	1	7
<i>Piping.</i> Aed. arg.	1	1
<i>Pit.</i> An. cost.	1	1
Cul. dec.	1	1
Aed. arg.	1	1
Cul. neb.	2	3
<i>Pools.</i> An. cost.	51	74	25	4	25	18	12	...	209
Aed. arg.	20	37	10	5	1	...	73
Cul. fat.	4	3	1	8
An. cost. and Cul. dec.	1	1
An. cost. and Aed. arg.	1	1	...	5
Cul. neb.	3	3	...	24
Cul. dec.	6	3	12	1
Cul. fat. and Cul. dec.	1	1

TABLE (12)—continued.

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
Pots. Aed. arg.	7	63	74	46	271	656	380	325	290	174	106	182	2,574
Cul. neb.	8	14	29	22	48	117	113	171	280	203	179	164	1,348
Aed. arg. and Cul. neb.	1	1	2	3	2	2	11	3	5	1	31
Cul. neb. and Cul. dutt.	1	2	3
Lutz. tigr.	1	1	...	2	1	5
Cul. fat.	1	7	13	6	10	15	6	3	2	63
Aed. apicoarg	1	1	2	4
An. cost. Aed. arg. and Cul. thal.	1	1
An. cost.	108	12	6	3	...	3	...	132
An. cost. Aed. arg. and Cul. dec.	1	1
Cul. fat. and Cul. neb.	2	...	1	4	7
An. cost. and Psych	1	1
An. cost. and Aed. arg.	1	3	5	9
An. cost. and Cul. neb.	1	1	2
Cul. fat. Cul. dec. and Aed. arg.	1	1
Aed. arg. and Cul. dec.	1	1	2
Cul. dutt.	7	...	2	38	7	5	...	59
Aed. arg. and Cul. dutt.	2	3	5	1	10
Aed. irr.	1
Cul. inv.	1	1	2
Cul. inv. and Cul. dutt.	1	1
Cul. dec.	4	3	1	1	...	9
Aed. arg. and Aed. lut.	1	2	3
Cul. dec. and Aed. lut.	1	1
Aed. lut.	1	1
Aed. arg. and Cul. fat.	2	5	1	1	5	...	1	...	15
Aed. apicoarg.	1	1
Snail shell. Aed. arg.	1	...	1
Cul. neb.	1	...	1
Tank. Aed. arg.	5	2	2	2	1	...	15
Tarpaulin. Aed. arg.	1	1
Tea-pot. Aed. arg.	1	1	2

TABLE (12)—continued.

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
<i>Tin.</i>													
Cul. neb.	—	1	—	—	7	19	10	3	30	22	25	13	130
Aed. arg.	—	—	2	2	30	70	32	10	14	19	15	10	204
Aed. arg. and Cul. neb.	—	—	1	—	...	1	—	—	1	—	1	—	4
Eret. chrys.	—	—	—	—	1	—	—	—	—	—	—	—	1
Cul. fat. and Aed. arg.	—	—	—	—	1	—	—	—	—	—	—	—	1
Cul. fat.	—	—	—	—	—	2	—	1	—	2	—	—	5
Aed. arg. and Aed. lut.	—	—	—	—	—	1	4	—	—	—	—	—	5
An. cost.	—	—	—	—	—	15	1	1	—	1	—	—	18
Aed. arg. and Cul. dec....	—	—	—	—	—	1	—	—	—	—	—	—	1
Cul. dutt.	—	—	—	—	—	—	2	—	—	—	—	—	2
Cul. inv.	—	—	—	—	—	—	1	—	—	—	—	—	1
Cul. dec. and Cul. neb.	—	—	—	—	—	—	1	—	—	—	—	—	1
Cul. dec.	—	—	—	—	—	—	—	1	1	—	—	—	2
Aed. lut.	—	—	—	—	—	—	—	—	—	1	1	—	2
<i>Trees, Paupaw.</i>													
Cul. neb.	—	—	—	—	4	—	7	—	—	—	—	—	11
Aed. arg.	—	—	—	—	5	—	2	—	—	—	—	—	7
Aed. lut.	—	—	—	—	1	—	1	—	—	—	—	—	2
An. cost.	—	—	—	—	1	—	—	—	—	—	—	—	1
<i>Banana.</i>													
Aed. arg.	—	—	—	—	5	10	26	—	—	—	—	—	41
Cul. neb.	—	—	—	—	1	1	2	—	—	—	—	—	4
Cul. fat.	—	—	—	—	—	—	1	—	—	—	—	—	1
Aed. lut.	—	—	—	—	—	—	1	—	—	—	—	—	1
Aed. arg. and Aed. lut.	—	—	—	—	1	—	—	—	—	—	—	—	1
<i>Mango.</i>													
Aed. lut	—	—	—	—	2	1	1	—	—	—	—	—	4
Aed. arg.	—	—	—	—	2	—	—	—	—	—	—	—	2
Aed. apicoan. and Aed. lut.	—	—	—	—	1	—	—	—	—	—	—	—	1
<i>Breadfruit.</i>													
Aed. lut.	—	—	—	—	2	1	3	—	—	—	—	—	6
Aed. arg.	—	—	—	—	—	—	2	—	—	—	—	—	2
<i>“Fruit tree.”</i>													
Aed. lut....	—	—	—	—	2	—	—	—	—	—	—	—	2
Aed. afric.	—	—	—	—	1	—	—	—	—	—	—	—	1
Aed. arg.	—	—	—	—	—	1	—	—	—	—	—	—	1

TABLE (12)—continued.

	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
<i>Cashew tree.</i> Aed. lut.	2	2
<i>Unnamed trees.</i> Aed. lut.	3	4	11	18
Aed. arg.	3	10	10	23
Cul. neb.	3	6	9
Cul. fat.	2	2
Aed. arg. and Cul. neb....	2	2
Aed. arg. and Aed. lut....	1	1	2
<i>Tyre.</i> Aed. arg.	5	10	4	...	2	1	1	...	23
Cul. neb.	3	5	2	1	...	11
An. cost.	1	1
<i>Well.</i> Aed. arg.	1	1	...	11	9	10	5	6	1	2	5	51
Cul. fat.	2	1	3
Cul. neb.	2	2	2	5	3	14
An. cost.	1	...	2	3
Cul. dec.	2	...	2
Cul. dutt.	1	1
Cul. fat. and Cul. neb.	2	2
<i>Marine Boats and Barges.</i>													
Aed. arg.	19	2	11	4	36
Cul. fat.	1	1
An. cost.	2	2
Cul. dec.	2	...	2	...	4
Cul. neb.	1	...	1
Cul. dec. and Cul. fat.	2	2
Cul. fat. and Aed. arg.	1	...	1
Totals ...	21	90	130	86	591	1,431	889	645	908	642	577	517	6,527

Each case is considered first from a clinical view-point, and the appearance, extent and probable duration of the lesion are noted. If thought necessary, cultures are made, the media employed being Sabouraud's maltose agar, glucose agar, and glucose hydrocele broth. Finally, a biopsy is made, unless the patient refuses. Freezing the part with Anestile is adopted as being the method least damaging to the tissues and most comfortable for the patient. Little difficulty has been experienced in gaining consent for this most important step in the examination, and the small wound, if kept clean, heals rapidly.

Photographs are also taken, whenever possible.

The patients are kept under observation, subsequent to their first appearance and the effect of treatment is noted. At the present stage of the investigation, it is not possible to give a classification or state conclusions. It is hoped, however, that when a sufficient number of lesions have been examined, enough experience will have been gained and a sufficiency of data gathered to allow of a grouping of similar types.

The various forms of ulcer promise in themselves an extensive field for study. So far two varieties have been identified and separated from the remainder. To one class belong those ulcers of a tubercular or lupoid character, in which sections show the presence of tubercles with giant cells and acid-fast bacilli. In another category are placed those of a mycotic origin. From the lesions in this type a yeast-like fungus has been recovered which has shown a certain amount of virulence to guinea-pigs. Other conditions such as Lichen pilaris, various forms of Tinea, Psoriasis and Impetigo have been diagnosed on histological and cultural findings.

HISTOLOGICAL.

Apart from specimens connected with yellow fever, which are considered under their appropriate heading, a considerable number of interesting tissues were received.

The specimens consisted mainly of tumours or parts of tumours. Malignant growths numbered twelve, comprising seven carcinomata and five sarcomata.

Details are as follows:—

Squamous carcinoma	Back.
"	"	...	Leg.
"	"	...	Gland.
Adeno carcinoma	Liver (two).
"	"	...	Ovary.
Papilloma	Skin.
Sarcoma round celled	Leg.
"	"	"	Hand.
"	"	"	Liver.
"	"	"	Eye-ball.
"	"	"	Testes.

Simple tumours numbered fourteen, as follows:—

Adenoma	Lip.
Fibroma	Foot.
Mixed tumour	Parotid.
Lymphangioma	Face.
Fibromyoma	Uterus.
Gumma	Abdomen.
Granuloma	Eye-ball (two).
Onchocerca	Abdomen.
Keloid	Neck.
Chronic Inflammatory	Finger.
"	"	...	Gland.
"	"	...	Scrotum.

Six specimens of spleen were received. Two showed cirrhosis, the spironema of relapsing fever was demonstrated in two, and tubercular changes were present in one.

There were also six specimens of liver. Cirrhotic changes were present in three, these being associated with the presence of encysted *Porocephalus* larvæ in one.

Four specimens of brain were examined. There was a large intraventricular hæmorrhage in one, and in another, from a native child, the capillaries showed numerous sporulating subtertian parasites.

Four specimens of kidney, two of stomach, one of skin and one of spinal cord showed nothing noteworthy.

There were four specimens of lung. Tubercular disease was present in two and gangrene in one.

Two specimens of bowel were received. One showed extensive ulceration due to *E. histolytica* and the other showed a perforation due to a foreign body.

The brain, or parts thereof, from three dogs was examined. Negri bodies were demonstrated in one animal, which had been shot at Warri. This last case forms the subject of a paper which awaits publication.

EXAMINATION OF DRIED SMEARS.

Ninety-three smears were received. Details are as follows:—

Bubo	24.	Bacillus pestis in	13.
Spleen	13.	„ „ „	2.
Lung	13.	„ „ „	2.
Liver	12.	„ „ „	2.
Heart	11.	„ „ „	1.
Penile sores	8.	Spirochaetes „	4.
Skin sores	4.	No Leishmania.	
Nasal	4.	No <i>B. leprae</i> .	
Throat	3.		
Prostatic	2.	Intra-cellular gram-negative diplococci.	
Cerebrospinal fluid	2.	One Pneumococcal.	One meningococcal.
Vaginal	1.		
Gland	1.	No tryps:	
Conjunctival	1.		
Abscess	1.		

EXAMINATION OF URINE.

Thirty samples were examined microscopically. Tube casts were found in nine. Pus occurred in one, and Schistosome ova in another. One sample was quantitatively analysed for sugar.

EXAMINATION OF SPUTUM.

Twenty-five specimens were examined for tubercle bacilli. These were present in three samples from Europeans and in two samples from Native Africans.

In addition to these, however, a number of sputa were examined for the presence of plague bacilli.

SEROLOGICAL.

Thirty-five Sachs-Georgi reactions were carried out. There was a positive result in five.

The Widal test was performed in twenty-two cases. There was a positive result with *Bacillus paratyphosus* B. in two cases, and to *Bacillus paratyphosus* C. in three cases.

MISCELLANEOUS.

A number of Vaccines were prepared for various diseases. A routine bacteriological analysis of the Lagos Water Supply was also done.

In addition to these, the blood of a certain number of horses was examined and trypanosomes were found in a small proportion.

The fæces of a number of dogs was examined and *Ankylostome* ova were found in practically all.

EXAMINATION OF FÆCES.

Rarely are specimens of fæces sent to the Research Institute from outstations.

Practically all samples came from Lagos and its environs, namely Ikoyi, Apapa, Iddo and Ebute Metta. The nearest of these to the Institute is three miles and the farthest is eight miles. Such distances mean time lost in transit, and the longer the period after voiding, the greater is the difficulty in obtaining correct findings from an examination of the stools. This was one of the strongest reasons for the establishment of a clinical laboratory within the precincts of the Hospital at Lagos.

During 1925, however, except in the first and the last quarters of the year, the clinical laboratory was without a pathologist, so that the number of examinations of fæces was restricted. Such specimens as were of diagnostic importance were sent to the Research Institute at Yaba.

The period covered by this review, therefore, is roughly eight months, during which 189 specimens were received.

As in previous Reports, a table has been constructed classifying the results, and on this occasion it is limited to samples from European sources, which numbered one hundred and sixty-one.

The stools, as received at the Research Institute were referred to three main groups, (i) Formed, (ii) Pultaceous and (iii) Liquid.

Each of these was again sub-divided according as mucus was present or absent.

Column I in the Table (Table 13) refers to formed stools. These amounted to thirty-two, a proportion of 19·87 per cent. Mucus was present in twelve and was not observed in twenty; there were positive findings in nine of the former and in only one of the latter.

Column II concerns pultaceous stools. Of this class there were seventy-eight, a proportion of 48·44 per cent. Mucus was noted in eight and was not seen in seventy; there was one negative finding in the former group and forty-nine in the latter.

Column III deals with liquid stools, which numbered fifty-one, a proportion of 31·67 per cent. All the specimens containing mucus, namely seventeen, gave positive findings contrasting with thirty-four showing no mucus, of which thirteen gave positive findings. It will be gathered, from these data that the stools of Europeans in Lagos are usually pultaceous.

It will also be observed that the presence of mucus is practically always indicative of a diseased condition of the large intestine.

Another interesting feature is the large proportion of negative findings in liquid motions which contain no mucus. In most of these

cases the bowel condition is an irritative one due to dietetic and other indiscretions, to unscund food and to a variety of other causes, and it clears up rapidly.

The absence of helminth ova was remarkable. In one case only were they found, and they were noted as *Taenia*.

Entamoeba histolytica either in the free or in the encysted stage was demonstrated in twenty-three cases; in sixteen instances the stools in which they occurred contained mucus.

It is necessary to consider the occurrence of twenty-seven cases which showed no pathogenic protozoa but which contained one or more of the following: red blood cells, pus cells, epithelial cells or Charcot-Leyden crystals. It is obvious that the length of time before the specimen came to the microscope, in many of these cases would allow of sufficient degeneration in the pathogenic *entamoeba* to render it unrecognisable or at least doubtful.

Again, in occasional instances the finding of only epithelial cells is not necessarily an indication of an ulcerated condition of the lower bowel. It has happened on several occasions that mucus is found in a stool from a case in which there was a previous history of dysentery, the mucus seemingly being the result of a simple irritative process imposed on a bowel which had not completely recovered its function.

But there remain cases in which the bacillary form of dysentery must be considered. With a view to determining whether the Flexner and the Shiga bacilli play an etiological role in the local form of dysentery, thirteen cases were selected, in which *amœbæ* were not definitely identified, but all of which showed evidence of ulceration in the form of red-blood cells and pus cells. Mucus and blood were noted macroscopically in ten and in the remaining three the microscope showed the presence of pus and epithelial cells.

A small piece of blood-tinged mucus or a drop of the liquid *fæces*, freshly passed, was picked up on a platinum loop and transferred to a tube of nutrient broth. Three or four hours later four McConkey plates were inoculated from the broth.

A non-lactose fermenter was recovered from five cases, in all of which there was blood and mucus in the stool.

A full history of one of these cases has already been published, (*Journal of Tropical Medicine and Hygiene*, Volume XXVIII, No. 21, November 2nd, 1925, pages 379-381).

This was the only case in which death occurred. All the others in the series improved rapidly while being treated with Emetine.

The sugar reactions and other characteristics of the non-lactose fermenting organisms are seen in Table 14.

It is difficult to state to what type or types the organisms belong. The important features are, the non-fermentation of lactose (even after several weeks), the absence of gas-formation, and the lack of motility.

In no case did a known type Flexner or type Shiga serum agglutinate the organism.

Further work requires to be done on these and on similar cases.

In pursuing this research one must bear in mind the conception of Dobell and O'Connor ("The Intestinal Protozoa of Man," London, 1921, page 41) that a state of equilibrium between *E. histolytica* and its human host must be regarded as a "normal" or "typical" condition, and that only when they do not live in harmony does a pathological condition result.

An invasion by bacilli, such as those isolated in the present series of cases, may be the determining factor in the onset of dysentery.

TABLE (13).

	Formed mucus.		Pulaceous mucus.		Liquid mucus.		Total.
	+	—	+	—	+	—	
E. histolytica, pus, epithelial and red blood cells	8	...	10
E. histolytica, pus and epithelial cells	1	...	2	...	2	2	8
E. histolytica, Trichomonas and Blastocystis	1	...	1
E. histolytica, and Charcot-Leyden crystals	1	1
E. histolytica, cysts, and Trichomonas	1	1
E. histolytica cysts and Blastocystis...	...	1	1
E. histolytica cysts	1	1
Charcot-Leyden crystals	1	1
Charcot-Leyden crystals and epithelial cells	1	1
Charcot-Leyden crystals, pus and epithelial cells	1	1
Pus, epithelial and red blood cells	2	...	2	...	4
Pus and epithelial cells	3	2	4	...	14
Epithelial cells	5	2	...	5	7
Blastocystis	3	...	3	6
Iodamœba butschlii and Blastocystis	1	1
Cercomonas and Blastocystis	2	2
Iodamœba butschlii, spirochaetes and Cercomonas	1	1
Iodamœba butschlii, Cercomonas and epithelial cells	1	1
Chilomastix	1	...	1	2
E. coli and E. nana	1	1
E. nana and Blastocystis	1	1
E. coli	1	1
Chilomastix and Blastocystis	3	...	1	49	...	1	1
Negative	21	93
Total	12	20	8	70	17	34	161

TABLE (14).

	Mannite.	Glucose.	Maltose.	Saccharose.	Galactose.	Arabinose.	Raffinose.	Sorbite.	Indol.	Motility.
O.	A	A	—	—	—	—	—	—	—	—
S.	—	A	—	—	—	—	—	—	—	—
E.	—	A	—	—	—	—	—	—	—	—
T.	—	A	—	—	—	—	—	—	—	—
C.	—	A		A	A	—	—	—	—	—

EXAMINATION OF BLOOD SMEARS.

A certain amount of routine clinical work which had to be done at different times during the year included one hundred and eighty blood smears. In addition ninety-four smears were received from outstations. No parasites were found in two hundred and thirty-six. Subtertian rings were present in twenty.

The spironema of relapsing fever was found in fifteen, all of these slides coming from outstations, principally in the north.

Trypanosomes were present in one smear sent from Cameroons. Embryos of *Filaria loa* were found in one specimen.

Pigmented mononuclears were noted in a smear which showed no malarial parasites.

The subtertian parasites were, with one exception, found in smears from Europeans. All the other parasites were found in smears from Africans, except in the case of *Filaria loa*.

There were two cases of Leukaemia in natives, an occurrence sufficiently unusual to merit record. The predominant leucocyte in one was polymorph and in the other the lymphocyte. In the latter instance Cancrum oris was present and a description of this case will appear in the Transactions of the Royal Society of Tropical Medicine, London.

Forty-five differential leucocyte counts were made, and the Arneth formula was ascertained in seventeen smears. These are detailed under the heading of "Blackwater Fever" and "Yellow Fever."

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